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Poultry as a Host for the Zoonotic Pathogen *Campylobacter jejuni*

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Abstract

Campylobacteriosis is the most reported foodborne gastroenteritic disease and poses a serious health burden in industrialized countries. Disease in humans is mainly caused by the zoonotic pathogen *Campylobacter jejuni*. Due to its wide-spread occurrence in the environment, the epidemiology of *Campylobacter* remains poorly understood. It is generally accepted, however, that chickens are a natural host for *Campylobacter jejuni*, and for *Campylobacter* spp. in general, and that colonized broiler chicks are the primary vector for transmitting this pathogen to humans. Several potential sources and vectors for transmitting *C. jejuni* to broiler flocks have been identified. Initially, one or a few broilers can become colonized at an age of >2 weeks until the end of rearing, after which the infection will rapidly spread throughout the entire flock. Such a flock is generally colonized until slaughter and infected birds carry a very high *C. jejuni* load in their gastrointestinal tract, especially the ceca. This eventually results in contaminated carcasses during processing, which can transmit this pathogen to humans. Recent genetic typing studies showed that chicken isolates can frequently be linked to human clinical cases of *Campylobacter* enteritis. However, despite the increasing evidence that the chicken reservoir is the number one risk factor for disease in humans, no effective strategy exists to reduce *Campylobacter* prevalence in poultry flocks, which can in part be explained by the incomplete understanding of the epidemiology of *C. jejuni* in broiler flocks. As a result, the number of human campylobacteriosis cases associated with the chicken vector remains strikingly high.

Key Words: Campylobacteriosis—*Campylobacter jejuni*—Chicken vector—Zoonosis.

Introduction

CAMPYLOBACTER INFECTIONS ARE NOW the leading cause of human bacterial gastroenteritis in many developed countries. Although the number of registered campylobacteriosis cases has declined slightly in some parts of the world during recent years, the overall disease burden is still noteworthy (Ailes et al. 2008, EFSA and ECDC 2011). The true incidence of campylobacteriosis in industrialized countries is uncertain since many unreported infections occur for every diagnosed case. In the United States, the Foodborne Diseases Active Surveillance Network (FoodNet) reported an incidence of culture-confirmed *Campylobacter* infections in the FoodNet sites of 12.7 per 100,000 persons in 2006 (Ailes et al. 2008). These numbers represent a 30% decline compared to

the 1996 situation, but the incidence still remains above the national health objective. Most other regions of the world report a higher disease incidence, with strikingly high numbers in New Zealand in 2003 of almost 400 cases per 100,000 people (Baker et al. 2007). Within the European Union campylobacteriosis has been the most frequently reported zoonotic disease in humans as from 2004. In 2009, the overall EU notification rate was 45.6 cases per 100,000 inhabitants (EFSA and ECDC 2011). *Campylobacter* enteritis in humans is mainly caused by *Campylobacter jejuni* (EFSA and ECDC 2011). In 2009, *C. jejuni* accounted for 90% of the cases characterized at species level. *Campylobacter coli*, *Campylobacter lari*, and *Campylobacter upsaliensis* accounted only for, respectively, 2.5%, 0.2%, and 0.01% of the isolates. The remainder of the speciated isolates included other (unknown) species. Regional

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or country differences, however, do exist: in Bosnia and Herzegovina, for instance, a higher prevalence of *C. coli* in sporadic human infections (30%) has been noted (Uzunović-Kamberović et al. 2007).

As will be further on discussed in this review, there is increasing evidence that poultry is the number one contributor to disease in humans. Nevertheless, no effective strategy exists to clear *Campylobacter* from broiler flocks (Hermans et al. 2011a). Although some reduction in the number of colonized flocks was observed upon implementing specific hygienic control strategies, as well as in bacterial counts from colonized birds upon therapeutic feeding with anti-*Campylobacter* substances, no commercial product is available that effectively prevents or reduces *Campylobacter* colonization of the avian gut. Therefore, the number of human campylobacteriosis cases remains strikingly high. In the next sections, the prevalence of *Campylobacter* in the environment and animal hosts will be discussed and poultry animals will be identified as a natural host for this zoonotic pathogen. These birds are under a constant contamination pressure and lots of risk factors can contribute to *Campylobacter* colonization in poultry, being in part responsible for the failure of current control measures. A thorough discussion of these risk factors will finally be followed by highlighting the poultry reservoir as the main contributor to campylobacteriosis in humans.

Campylobacter Prevalence in Animals and the Environment

Paradoxically, despite its fastidious, fragile nature, *Campylobacter* is highly prevalent in the environment and can survive for prolonged periods both inside and outside a suitable host (Newell 2002, Murphy et al. 2006). How the microorganism copes with stresses encountered in the environment still remains enigmatic, but, clearly, *Campylobacter* developed some survival mechanisms to overcome these stressors (Murphy et al. 2006). The presence of highly mutable sites in the *C. jejuni* genome is responsible for its rapid adaptation in a novel host (Jerome et al. 2011). *Campylobacter* can be frequently found in surface water and is part of the natural intestinal microbiota of a wide range of wild and domestic animals, especially poultry (Newell 2002, Whyte et al. 2004, Abulreesh et al. 2006, Young et al. 2007, Ogden et al. 2009, Jokinen et al. 2011). The estimated *Campylobacter* prevalence in poultry and nonpoultry farm animals depends on season, age of animal, flock or herd size and type, diet, husbandry practices, and geography, with *C. jejuni* being the most isolated species (Kuana et al. 2008, McDowell et al. 2008, Zweifel et al. 2008, Ellis-Iversen et al. 2009, Messens et al. 2009, Näther et al. 2009, EFSA 2010a, EFSA and ECDC 2011, Jorgensen et al. 2011). In pigs *C. coli*, however, dominates (84% of the isolates in 2009) (EFSA and ECDC 2011). Surprisingly, in some countries, like Spain, Luxembourg, Slovenia, and Bosnia and Herzegovina, a much higher proportion of *C. coli* compared to *C. jejuni* was also isolated from poultry samples (Zorman et al. 2006, EFSA and ECDC 2011), which is clearly, at least in part, responsible for the higher prevalence of *C. coli* in sporadic human infections in these latter two countries (see above).

Poultry as a Natural Host for Zoonotic Campylobacter

Chickens are a natural host for thermotolerant or thermophilic *Campylobacter* species (containing the above-mentioned

species and for simplicity hereafter referred to as *Campylobacter*) (EFSA and ECDC 2011). The probability of a flock to become colonized increases during rearing, resulting in on average 60%–80% of the analyzed broiler flocks to be positive for *Campylobacter* species in general at slaughter age worldwide (Herman et al. 2003, Rasschaert et al. 2006, Kuana et al. 2008, Reich et al. 2008, EFSA 2010a). Striking differences in EU prevalence do exist. In 2008, almost all broiler batches in some Northern European countries, like Estonia, Norway, and Finland, were free of *Campylobacter*, whereas a strikingly high prevalence of 100% was reported for Luxembourg. In general, *C. jejuni* was found to be the dominating species. Of the analyzed flocks on average 41% were colonized specifically with *C. jejuni* in the EU and even up to 55% in Brazil. Colonized poultry flocks might contaminate the surrounding environment by which *Campylobacter* is able to spread further and contaminate other farms or humans (Jonsson et al. 2010).

Colonization of Broiler Chickens by *C. jejuni* at the Farm

From day-of-hatch until the broiler chickens are transported to the abattoir, the animals can encounter several risk factors contributing to their colonization with *C. jejuni*. As a consequence, the *Campylobacter* ecology and epidemiology in broiler flocks is quite complex. An overview of the risk factors involved in the environmental transmission of *Campylobacter* to broiler flocks is given in Table 1.

Initial broiler flock colonization and colonization pattern

The cecum is the predominant site for colonization, where the organism resides principally in the mucus layer of cecal crypts (Beery et al. 1988, Meade et al. 2009). *C. jejuni*-colonized broiler chickens carry high bacterial numbers in their ceca (generally around 10^6 to 10^8 cfu/g) and remain colonized until slaughter (Evans 1992, Jacobs-Reitsma et al. 1995, Evans and Sayers 2000, Allen et al. 2008, Stern 2008). Bacterial numbers of up to 10^{12} cfu/g have been isolated from chicken ceca after oral challenge with *C. jejuni* (Meade et al. 2009). Upon colonization with *Campylobacter*, the chick immune system is only activated inefficiently and expression of several antimicrobial peptide genes is reduced, which both contribute to the persistent high-level commensal colonization of *Campylobacter* in the avian gut (Meade et al. 2009, Hermans et al. 2011b).

Initial colonization of broiler chickens probably occurs through horizontal transmission from the environment, whereas vertical transmission from breeder hens or carryover of infection from a positive flock to a new flock in the same house, after cleansing and disinfection, are considered to be unlikely (van de Giessen et al. 1992, Jacobs-Reitsma et al. 1995, Bull et al. 2006, Patriarchi et al. 2011). Indeed, carryover of *C. jejuni* subtypes between broiler flocks in the same house seems to occur only rarely (Barrios et al. 2006, Colles et al. 2008, McDowell et al. 2008). Persistent clones in the outside environment can, however, be responsible for repeated infection of multiple broiler flock rotations (Petersen and Wedderkopp 2001, Wedderkopp et al. 2003). Some *C. jejuni* strains can be very persistent in a confined geographical area. In a Lithuanian study, a single amplified fragment length polymorphism type was found in several broiler farms over a 1-year period (Kudirkiene et al. 2010).

Colonization of broiler chickens with *C. jejuni* is influenced by many factors, including source of the microorganism, the

TABLE 1. RISK FACTORS FOR *CAMPYLOBACTER* COLONIZATION OF BROILER FLOCKS AT THE FARM

<i>Rearing stage</i>	<i>Influencing factor</i>	<i>Risk factor</i>	<i>Reference</i>
Initial colonization	Source of the microorganism and infective dose	Persistent clones in the environment, high colonizer strains and high infective dose	Stas et al. (1999) Petersen and Wedderkopp (2001) Wedderkopp et al. (2003) Kudirkiene et al. (2010)
	Age of the animals Flock size	From 2 weeks onward Higher slaughter age Increased flock size	Jacobs-Reitsma et al. (1995) Herman et al. (2003) van Gerwe et al. (2009) Barrios et al. (2006) Berndtson et al. (1996b) Barrios et al. (2006)
	Seasonality	Summer months	McDowell et al. (2008) Ellis-Iversen et al. (2009) Ellerbroek et al. (2010) Jorgensen et al. (2011)
	Applied husbandry practices	Rainfall Ineffective hygiene measures	Jorgensen et al. (2011) McDowell et al. (2008) Allen et al. (2011)
	Sources for horizontal transmission	Other colonized animals on the farm Rodent and insect carriers	van de Giessen et al. (1996) Ridley et al. (2008) Zweifel et al. (2008) Ellis-Iversen et al. (2009) Hanel et al. (2009) Allen et al. (2011) Patriarchi et al. (2011) Ridley et al. (2011) Hald et al. (2004) Nichols (2005) Hald et al. (2008) Hazeleger et al. (2008) McDowell et al. (2008)
		Contaminated surface water Personnel and farm equipment	Messens et al. (2009) Ramabu et al. (2004) McDowell et al. (2008) Ridley et al. (2011b)
		Partial depopulation?	Allen et al. (2008) Patriarchi et al. (2011) vs. Russa et al. (2005) Barrios et al. (2006) Näther et al. (2009)
Transmission through the flock	Bird-to-bird transmission by fecal-oral route	Drinking water and feed	Evans (1992) Gregory et al. (1997) Herman et al. (2003) Gellynck et al. (2008) Messens et al. (2009) Sparks (2009)
Colonization pattern	Increased intestinal bacterial load	Absence of anti- <i>Campylobacter</i> substances Chicken diet	Connerton et al. (2004) El-Shibiny et al. (2005) Udayamputhoor et al. (2003)
Transportation	Increased intestinal bacterial load and fecal <i>Campylobacter</i> excretion rates	Transport-induced stress Contaminated transport crates	Stern et al. (1995) Whyte et al. (2001) Slader et al. (2002) Herman et al. (2003) Hansson et al. (2005) Rasschaert et al. (2007) Hastings et al. (2010) Patriarchi et al. (2011)

infecting dose, and age of the animal (Stas et al. 1999). Most flocks become colonized only at an age of 2–4 weeks (Jacobs-Reitsma et al. 1995, Berndtson et al. 1996a, Herman et al. 2003, van Gerwe et al. 2009). Protection of young chickens against colonization can be attributed to *Campylobacter*-specific maternal antibodies (Sahin et al. 2003), the titer of which generally drops after 2 weeks (Cawthraw et al. 1994). As a consequence, after this protection period animals are more susceptible to colonization with *Campylobacter* and this susceptibility increases with higher slaughter age (Berndtson et al. 1996b, Barrios et al. 2006). Although during colonization antibodies specifically directed against *Campylobacter* are induced (Cawthraw et al. 1994), the bacterium is apparently not expelled from its host. Also, the time of year clearly has an influence. In Germany and the United Kingdom, the risk for broilers to become colonized with *Campylobacter* is highest during the summer months (McDowell et al. 2008, Ellis-Iversen et al. 2009, Ellerbroek et al. 2010, Jorgensen et al. 2011) and a coincident seasonality of infections in chickens and humans has been shown (Meldrum et al. 2005). In the EU-wide baseline survey on *Campylobacter* in broiler batches in 2008, batches were most likely to be found *Campylobacter* colonized in the third quarter (July–September) of the year (EFSA 2010b). Very recently, a significant relationship was observed between several climatic factors (such as environmental temperature and amount of sunshine and rainfall) and *Campylobacter* prevalence in United Kingdom broiler flocks before first partial or full depopulation (Jorgensen et al. 2011), which could explain this observed seasonality. Seasonality/temperature, however, explained only half of the *Campylobacter* prevalence, indicating that also other factors such as husbandry practices and biosecurity may also be important. Indeed, a recent study by Allen et al. (2011) in the United Kingdom showed that the colonization of organic flocks is largely dependent on the husbandry practices used on the farm. In contrast, the length of the rearing period did not seem to have a large influence on *Campylobacter* prevalence. Next, an increased flock size was associated with a higher probability of that flock to be colonized with *Campylobacter* (Berndtson et al. 1996b, Barrios et al. 2006). Finally, in commercial flocks, the appearance of *Campylobacter*-specific bacteriophages and naturally occurring anti-*Campylobacter* substances have been associated with changes in level of colonization (Connerton et al. 2004, El-Shibiny et al. 2005), and cecal colonization of birds receiving plant-protein-based feed is significantly lower compared to that of birds receiving animal-protein-based feed or a combination of these two protein sources (Udayamputhoor et al. 2003).

Sources for horizontal *Campylobacter* transmission to broiler flocks

Colonized livestock and free-living animals are an important risk factor for transmitting *C. jejuni* to broiler flocks as *C. jejuni* genotypes from cattle, pigs, and laying hens, present at poultry farms, can also be found in the broiler flocks (van de Giessen et al. 1996, Ridley et al. 2008, Zweifel et al. 2008, Ellis-Iversen et al. 2009, Allen et al. 2011). Bovine *Campylobacter* isolates are indeed able to efficiently colonize chickens (Hanel et al. 2009) and might thus be a source for broiler chicken infection and human disease, although they differ significantly from chicken and human isolates by a reduced prevalence

of two genetic markers especially, the *dmsA* and γ -glutamate transpeptidase (*ggt*) gene, important for persistent colonization of *C. jejuni* in chickens (Barnes et al. 2007, Gonzalez et al. 2009). Probably, only bovine clones carrying *ggt* are able to efficiently colonize chicks and are thus a possible source for the transmission of *C. jejuni* to broiler flocks, but this hypothesis has yet to be confirmed. Whether *ggt*-negative *C. jejuni* clones are capable to acquire this marker through genetic exchange with *ggt*-positive clones, thereby promoting its successful colonization in the chicken gut, has yet to be examined. Also, several other studies revealed the presence of identical *C. jejuni* clones in bovines, chickens, and humans (Nielsen et al. 1997, On et al. 1998, Gilpin et al. 2008, Ragimbeau et al. 2008, Hakkinen et al. 2009, Huang et al. 2009), and even turkeys, sheep, water, dogs, and ostriches (Siemer et al. 2004). Recently, it was shown by molecular typing that *Campylobacter* strains from a broiler house and from an adjacent dairy farm were similar to those subsequently detected in the flock, indicating the importance of horizontal transmission and the risk of transmission of *Campylobacter* on multispecies farms (Ridley et al. 2011). This study also indicated that bovine fecal *Campylobacter* strains can colonize chickens, which was confirmed later on by Patriarchi et al. (2011). On a German farm, indistinguishable isolates of clonal origin were found in different flocks during the same rearing period (Ellerbroek et al. 2010). This suggests that *Campylobacter* strains might be transmitted from one broiler flock to another or might point toward a common external source infecting multiple broiler flocks at the same farm.

Also rodents, flies, and their larvae are potential vectors for *C. jejuni* transmission to broiler flocks (Berndtson et al. 1996a, Hald et al. 2004, 2008, Nichols 2005, Hazeleger et al. 2008). The importance of rodents as a potential vector is, however, somewhat controversial. Messens et al. (2009) stated that rodents should not be considered as a significant risk factor for the introduction of *Campylobacter* in broiler houses due to frequently applied on-farm rodent control programs. Meerburg (2010), on the other hand, stated that these programs are often poorly operated and mainly applied for economic, rather than for food safety purposes. Indeed, an association has been shown between the presence of rodents on farms and an increased risk for flocks to become infected with *Campylobacter* (McDowell et al. 2008), supporting Meerburg's hypothesis.

Another important source of infection is contaminated surface water as genotypes found in broilers can sometimes be detected in water puddles and ditch water as well, before the flocks are colonized (Bull et al. 2006, Messens et al. 2009). *C. jejuni* survival in water is promoted by several factors, including biofilm formation and possibly the viable but nonculturable state, in which *C. jejuni* enters when outside a suitable host (Sparks 2009). Biofilm formation has, however, been associated with decreased colonization potential in 1-day-old broiler chicks (Hanning et al. 2009). Viable but nonculturable *C. jejuni* cells are not believed to have reduced ability to attach to surfaces and once attached they may persist undetectable and be introduced into the food chain as soon as they come into contact with animals or products (Duffy and Dykes 2009).

Personnel and farm equipment such as trucks, forklifts, pallets, crates, and footwear have also been identified as potential sources of *C. jejuni* infection of broilers (Ramabu et al. 2004). Farm vehicles are often contaminated with

Campylobacter even after cleaning (Ridley et al. 2011b). Also, broiler flocks on farms with three or more broiler houses, low frequency of footbath disinfectant change, and decreased cleanliness of the broiler house ante-room have an increased risk to become colonized (McDowell et al. 2008).

Finally, partial depopulation/thinning of broiler flocks (early removal of a part of the birds) has been implicated as a potential risk factor for *Campylobacter* colonization of the remainder of the animals of these flocks due to difficulties in maintaining biosecurity during thinning (Patriarchi et al. 2011). Allen et al. (2008) observed an association between *C. jejuni* genotypes present on vehicles and crates arriving on a farm at thinning, and those subsequently recovered from the birds after slaughter. In addition, during this process particular *C. jejuni* strains were able to spread from one farm to another (nearby farm) when sharing the same bird-catching personnel and/or vehicles. However, in several other studies, no increased risk associated with this process has been shown (Russa et al. 2005, Barrios et al. 2006, Näther et al. 2009).

Transmission through the flock

Once flock colonization is detected, bird-to-bird transmission within flocks is very rapid. A recent mathematical model revealed a transmission rate of 2.37 (van Gerwe et al. 2009) new cases per colonized chick per day. This implies that in a flock of 20,000 broilers, the prevalence of *Campylobacter* would increase from one infected bird to 95% within the week after the first bird is infected. Indeed, in a study by Stern et al. (2001), the majority (95%–100%) of birds in a flock were colonized within 7 days after contact with a single (*Campylobacter*-colonized) seeder bird, regardless of the age of the animals. Drinking water and feed are believed to play an important role in the fecal–oral spread through the flock (Evans 1992, Gregory et al. 1997, Herman et al. 2003, Newell and Fearnley 2003, Sparks 2009). Once a flock is colonized, the nipple water is often contaminated with *C. jejuni* strains that are indistinguishable from those isolated from the broilers, indicating the importance of drinking water in transmitting this zoonotic pathogen throughout the flock (Gellynck et al. 2008, Messens et al. 2009).

The effect of transportation

Transport-induced stress has been shown to increase both the *Campylobacter* load (by 0.7 log₁₀ cfu/g) in broiler ceca (Stern et al. 1995) as well as its excretion rates in broiler feces (by 0.8 log₁₀ cfu/g) after transport (Whyte et al. 2001). Neither transportation distance nor duration significantly influenced the rate of *Campylobacter* shedding (Stern et al. 1995, Whyte et al. 2001). Transport crates are often still contaminated with *Campylobacter* when reused because crate decontamination processes are mostly ineffective (Ridley et al. 2011b). Contaminated crates can lead to external contamination of birds at partial thinning of the flock and during transport of a negative flock to the processing plant (Slader et al. 2002, Herman et al. 2003, Hansson et al. 2005, Rasschaert et al. 2007, Ridley et al. 2011b). However, there is still controversy about the possible role of transport crates in transmission of *Campylobacter*. Evidence for intestinal (co-)colonization due to transport in *Campylobacter*-contaminated containers was not found (Rasschaert et al. 2007). On the other hand, *C. jejuni* genotypes commonly associated with chickens were dominantly found

on transport equipment and persisted throughout the decontamination process, indicating that improperly disinfected transport crates could be involved in *Campylobacter* contamination of poultry flocks (Hastings et al. 2010, Patriarchi et al. 2011).

Carcass Contamination

A significant correlation exists between the *Campylobacter* colonization rate of broiler chickens during rearing and bacterial counts on their carcasses after processing (Herman et al. 2003, Rasschaert et al. 2006, Rosenquist et al. 2006, Reich et al. 2008). Worldwide, an average prevalence of *Campylobacter*-contaminated poultry carcasses is reported to be in the range of 60%–80% (Suzuki and Yamamoto 2009, EFSA 2010c, Mullner et al. 2010). Similarly as in live animals of a poultry flock, also on poultry carcasses *C. jejuni* is the predominating species (Rasschaert et al. 2006, Kuana et al. 2008, Suzuki and Yamamoto 2009, EFSA 2010a, 2010c). Almost all parts of contaminated carcasses, whether fresh, chilled, or frozen, are frequently contaminated with *Campylobacter* and are all likely to be important sources for disease in humans (Berndtson et al. 1992). Carcass contamination occurs during defeathering and evisceration, by contaminated feces leaking from the cloaca and visceral rupture of ceca carrying a high *Campylobacter* load (Berrang et al. 2001).

The source of the majority of *Campylobacter* genotypes contaminating a flock during the slaughter process is probably the live flock (Herman et al. 2003, Rosenquist et al. 2003, Colles et al. 2010). Indeed, colonized broiler batches are far more likely to result in contaminated carcasses after processing compared to *Campylobacter*-free batches (EFSA 2010b). Moreover, *Campylobacter* isolates found in the ceca of broilers are very often similar to those isolated from the corresponding carcasses (Normand et al. 2008). There is, however, evidence that the slaughter process increases the diversity of *Campylobacter* genotypes isolated from a flock (Colles et al. 2010), suggesting that other sources are involved as well. Indeed, carcasses can also become contaminated by cross-contamination of *Campylobacter* between birds within a flock and between flocks slaughtered successively (Herman et al. 2003, Rosenquist et al. 2003, Rasschaert et al. 2006) and even between flocks slaughtered in the same area, as evidenced in Quebec by Normand et al. (2008). *Campylobacter*-colonized flocks contaminate the abattoir environment upon entering the room and as a consequence *Campylobacter* can be isolated at all stages of the processing line (Ellerbroek et al. 2010). *C. jejuni* is able to survive overnight on these processing equipment surfaces, even after cleaning and disinfection (Peyrat et al. 2008). Therefore, surviving strains might possibly be a source of poultry carcass contamination of subsequent flocks, probably by intestinal contents of previously processed *Campylobacter*-colonized flocks (Newell et al. 2001, Miwa et al. 2003).

Combined hygienic approach to reduce *Campylobacter* prevalence in poultry

It is clear that lots of risk factors are involved in the environmental transmission of *Campylobacter* to broiler flocks (Table 1). Possibly, these factors are intimately linked with each other. The increased temperature during summer months for instance could promote the presence of flies

and rodents at the farm, whereas increased rain fall can create water puddle reservoirs in which *C. jejuni* can persist and transmit to other vectors (Jorgensen et al. 2011). Therefore, source attribution for *Campylobacter* colonization in poultry flocks is not straightforward and only a combined approach of properly implemented hygienic measures in all of these areas will be capable to significantly reduce the number of *Campylobacter* colonized flocks. Indeed, intensive on-farm cleaning procedures in the UK did reduce *Campylobacter* prevalence on broiler-harvesting equipment, vehicles and personnel but failed to reduce *Campylobacter* colonization of broiler flocks (Ridley et al. 2011b). This combined approach must aim at minimizing the probability that *Campylobacter* enters the broiler room (by rodent and insect control, foodbath disinfection for personnel working in the broiler room, and drinking water treatment), reduce bird-to-bird transmission (drinking water treatment), and prevent cross-contamination during transport (by decontamination of transport crates) and slaughter. Together with measures taken at retail, as well as consumer information campaigns, such an approach led to a 74% decrease in human campylobacteriosis cases attributed to poultry in New Zealand in 2008, resulting in a 54% decline in the overall notification rate for this country in 2008 compared to the 2002 to 2006 situation (Sears et al. 2011; see above). Because detailed data on *Campylobacter* prevalence in poultry flocks during this time course are not available, it can only be speculated that the decline in human illness in 2008 was due to a reduced prevalence of pathogenic *C. jejuni* in poultry that year.

Transmission to Humans

Transmission to humans most commonly occurs through consumption and handling of all kinds of foods of animal origin of which the carcasses are contaminated by *Campylobacter* during slaughter and carcass processing (Berrang et al. 2001, EFSA 2010c). In industrialized countries, handling, preparation, and consumption of contaminated chicken meat is considered to be the main source of infection in humans (Berndtson et al. 1992, Friedman et al. 2004). However, regular consumption of chicken meat reduces the risk for illness associated with recent chicken consumption, suggesting that partial immunity, conferring protection against *Campylobacter*, could be developed (Tam et al. 2009). By using genetic typing methods (see further) it was evidenced that chicken meat *Campylobacter* isolates can frequently be linked to human cases of campylobacteriosis. However, the overall genotypic diversity between isolates indicates that there are other sources contributing to disease in humans as well (de Haan et al. 2010, Thakur et al. 2010). Indeed, besides poultry, also nonpoultry farm animals can contribute to campylobacteriosis in humans. Not only does *Campylobacter* colonization of such animals pose a risk of contamination of their carcasses at slaughter, but it can also lead to the contamination of milk and surface water at the farm, as well as colonization of broiler flocks present at these farms (Doyle and Roman 1982, Stanley and Jones 2003, Garrett et al. 2006, Hannon et al. 2009), which are all risk factors for transmitting *C. jejuni* to humans. Also, direct contact with cattle, but also pets, in particular puppies with diarrhea, is a possible route of contamination (Tenkate and Stafford 2001, de Haan et al. 2010). Cattle and their direct

environment are thus potential reservoirs for zoonotic *C. jejuni* strains (Kwan et al. 2008). Next, also drinking water has been implicated as a possible source for human illness, although in the developed world waterborne infection of *Campylobacter* in humans is not very likely (Young et al. 2007). Finally, also raw vegetables, which can become contaminated by cross-contamination by other contaminated food products during preparation, but also directly at the farm, are an important source (Gardner et al. 2011, Verhoeff-Bakkenes et al. 2011), and were suggested to be the second highest risk factor, after handling and consumption of contaminated chicken products (Evans et al. 2003).

Contribution of the Chicken Reservoir to Campylobacteriosis in Humans

Due to the wide-spread occurrence of *Campylobacter* spp. their environmental cycle is not very well understood. Moreover, due to the possibility of cross-contamination, tracing the genuine source of *Campylobacter* infections is not straightforward. By using a genetic approach, however, Wilson et al. (2008) estimated that 97% of the number of sporadic human campylobacteriosis cases in England is attributable to animals farmed for meat, with chicken and cattle as main sources for *C. jejuni*. Wild animal and environmental sources would only contribute for 3% of the cases. These results indicate that contaminated food products are the principle source for disease in humans. Stern and Kazmi (1989) stated already over two decades ago that a large number of *C. jejuni* serotypes from poultry can frequently be linked to human cases of campylobacteriosis. This was confirmed by later reports using also genetic typing techniques (Zorman et al. 2006, Gonzalez et al. 2009, Mullner et al. 2010). In 1997 in Denmark, 36% of broilers were colonized by *C. jejuni* and a large overlap of the most common serotypes in humans and broiler chickens was shown (Nielsen et al. 1997). Biotyping and pulsed-field gel electrophoresis analysis revealed that ~20% of human *Campylobacter* isolates were genetically related to poultry isolates (Nadeau et al. 2002). Colles et al. (2008) found a significant similarity between chicken meat and human disease *C. jejuni* isolates. Comparison of *Campylobacter* *fla*-SVR genotypes isolated from humans and poultry revealed few significant differences in the distribution of genotypes over these two hosts (Wassenaar et al. 2009). Lindmark et al. (2009) detected a significant correlation between the presence of a particular *C. jejuni* subtype in patients and the consumption of fresh poultry meat during the same period and within the same geographical area. Together, all these observations indicate that certain *C. jejuni* strains circulate between poultry and humans, highlighting poultry as an important source for transmitting this pathogen to humans. This hypothesis is also strengthened by the observation that during the Belgian dioxin crisis in 1999, a withdrawal of chicken meat from the market in June coincided with a 40% decrease in human *Campylobacter* infections during that month (Vellinga and Van Loock 2002). Very recently, evidence of transmission by direct contact with poultry carcasses was given by Friis et al. (2010), who isolated identical strains from a poultry abattoir and a person that had developed campylobacteriosis upon entering that abattoir. Thus, there is increasing evidence that the broiler chick is a major reservoir for *C. jejuni* pathogenic to humans and that broiler chicken

meat contaminated with this zoonotic pathogen is the most important source for disease. For the European situation it was estimated that *Campylobacter*-contaminated chicken meat would be responsible for up to 40% of human campylobacteriosis cases. The chicken reservoir as a whole might even be responsible for up to 80% of the cases, because strains from the chicken reservoir may reach humans by pathways other than food (EFSA 2010c). As a consequence, eradicating *Campylobacter* from poultry lines could tremendously reduce *Campylobacter* enteritis in humans. Unfortunately, no effective, reliable intervention measure is available to date to reduce *Campylobacter* colonization in poultry (Hermans et al. 2011a). Neither the overall prevalence of this pathogen in chicken retail products, nor the number of reported poultry meat consumption-related human campylobacteriosis cases were reduced in recent years (EFSA 2010c, EFSA and ECDC 2011).

Concluding Remarks

Campylobacter occurs wide-spread in the environment and several vectors are able to transmit this pathogen to humans. Additionally, cross-contamination of several of these sources can occur, making genuine source attribution of campylobacteriosis cases in humans not straightforward. However, increasing evidence is available that the chicken reservoir is the main vector for *Campylobacter* transmission to humans. Chickens are a natural host for zoonotic *Campylobacter* species and broilers carry high bacterial numbers in their ceca until slaughter. This eventually results in contamination of their carcass in the abattoir. Recent reports using genetic typing methods proved that the poultry vector is indeed capable of transmitting this pathogen to humans. Despite the increasing evidence that poultry is the number one contributor to disease in humans, no effective strategy exists to reduce the *Campylobacter* prevalence in broiler flocks. As a consequence, the incidence of campylobacteriosis in humans remains strikingly high. A better insight into the *Campylobacter* ecology and epidemiology in chicks must therefore help to identify the rearing stages where the zoonotic pathogen can be introduced or persist in a flock. By focussing on these critical points it could be possible to develop intervention measures that are capable to minimize the number of colonized flocks and contaminated carcasses, and thus the number of human campylobacteriosis cases associated with the chicken vector. In the mean time it is of utmost importance that hygienic measures are properly implemented at all stages during rearing, forming a combined approach to limit *Campylobacter* entrance into a flock.

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